CME and the Medical Schools

MEDICAL SCHOOLS throughout the nation are beginning to assess, or reassess, their role and responsibility for the education of practitioners—or what is more commonly called continuing medical education (CME). This reevaluation is coming about partly as a result of a growing recognition that practitioner education is indeed part of a continuum of medical education which begins in medical school — or before — and continues through a lifetime of professional practice. But also it is occurring because of increasing professional and public concern with the continuing competence of practicing physicians in these times of rapid progress in medical science and rapid change in medical practice. Both the administration and the faculties of medical schools are becoming increasingly aware of the extent of the contribution of medical schools and their faculties to CME, whether under the auspices of the medical schools or not. And the fact that at least 16 states now require CME for physician relicensure has drawn the attention of the medical school faculties in those states to CME in a very personal way.

In California the Medical Injury Compensation Reform Act of 1975 (AB 1xx) requires that physicians have continuing medical education as a requirement for relicensure. In the wording of this law, continuing medical education is linked to continuing professional competence, to the physician's individual practice profile and to quality review, which in turn is again linked to CME. If one thinks about what these words mean and what these linkages entail, one senses that the enactment of this law may mark the beginning of a new era for CME. It asks CME to do some things it does not yet know how to do, but must learn to do, if it is to fulfill the public expectation of quality assurance in a physician's practice.

There are many actors on the CME stage. In the center as the main focus are the practicing physicians. But also on stage are professional associations, specialty societies, the public in the form of licensing boards, and medical school faculty members. The medical schools themselves may be thought of at present as being somewhere in the wings, perhaps getting ready to come on stage. It is noteworthy that each of the actors plays a lead role during some part of the CME scenario.

It seems likely that medical schools will soon come onto this stage and that they too will have a lead part to play. It seems certain that the role will require greater collaboration with the other actors than has been the case to date. Medical schools seem particularly qualified to help determine what new knowledge should be incorporated into practice, what is obsolete and should be discarded, and what remains current and should be affirmed. Medical schools can contribute a great deal to a better understanding and definition of physician competence, to the development of better educational methods and to research in quality assurance. But each of these will require substantial collaboration and interplay with the other actors on the CME stage.

In a sense, in developing their role in CME the medical schools may become like Janus, looking two ways at once—on the one hand studying and teaching in the world of science, and on the other studying and teaching in the world of practice. A great spin-off would be that practitioner education would affect undergraduate education as well as vice versa, with CME the conduit in both directions.

—MSMW

Management of Diabetic Ketoacidosis in Children

DEATH IN CHILDREN AND ADOLESCENTS with diabetic ketoacidosis is uncommon, provided early and appropriate treatment is given. Mortality in children has been attributed to cerebral edema[1,2] and inadequate replacement of potassium deficits.[3] One approach to reduce mortality is a modification of insulin dosage. Claims have also been made that morbidity of such patients is also lessened.

As reported in this issue by Kaufman and co-workers, the use of small doses of insulin, which are administered by continuous intravenous infusion, has achieved impressive results in the treatment of diabetic ketoacidosis in children[4-7]

Kaufman and co-workers, and other proponents of low-dose insulin, claim that its major benefits

are the minimized risks of hypoglycemia, hypokalemia and cerebral edema. The simplicity and safety of continuous intravenous infusion of small amounts of insulin and, theoretically, the attainment of physiologic blood levels of insulin have also been suggested as additional advantages of this treatment. It has been argued,[8,9] however, that although the serum concentrations of insulin resulting from the administration of "large" doses of insulin may exceed the levels required by peripheral tissues of normal persons for maximal insulin action, the attainment of supranormal blood insulin levels may be necessary to reverse increased hepatic gluconeogenesis and ketone production of diabetic ketoacidosis because the concentration of insulin normally perfusing the liver is substantially greater than that perfusing peripheral tissues.

In more than 1,800 patients with diabetic ketoacidosis admitted to the Childrens Hospital of Los Angeles (CHLA) since 1959, and treated with "large" doses of insulin,[10] the mortality approximated 0.2 percent. Because our results have been extremely satisfactory, we continue to use "large" doses of insulin (0.25 to 2 units per kg of body weight) subcutaneously as the initial dose, based on the degree of ketoacidosis and the blood glucose concentration.[10]

Although only a relatively few cases of children successfully treated with the small-dose continuous insulin infusion method have been described,[4-7] the results suggest that "small" doses of insulin may be equally effective as "large" doses. In comparative studies using different insulin regimens, small intravenous doses of insulin did not appear to be superior to conventional treatment with large doses.[7,11] In addition, the mode of administration of insulin probably does not make a significant difference in the outcome of therapy in most children and adolescents with diabetic ketoacidosis.

Factors other than insulin dose and method of administration that are essential to successful treatment include a physician experienced in the care of seriously ill diabetic children, prompt and appropriate replacement of fluid and electrolyte deficits, and making individual treatment adjustments after careful and close monitoring of the patients' clinical status.

Dehydration is always present in patients with moderate to severe diabetic ketoacidosis. In addition to requirements for maintenance fluid and electrolytes, fluid replacement should be carried out according to the degree of dehydration.[10] Hypokalemia may occur during treatment of diabetic ketoacidosis if potassium is not administered intravenously. In a recent study, potassium requirements were identical whether low-dose or large insulin doses were given.[11] All patients have depleted body stores of potassium and should receive early and continuous potassium replacement at a minimal rate of 3 mEq per kg of body weight per 24 hours, given intravenously. In some patients, particularly in those with a low serum potassium concentration (that is, $K_s < 4.0$ mEq per liter) on admission, the infusion of large amounts of potassium has been lifesaving.[10] In instances when the rate of potassium administration recommended above is exceeded, continuous electrocardiographic (EKG) monitoring should be used to observe for signs of potassium toxicity.

Complications secondary to the administration of sodium bicarbonate cause greater problems in diabetic patients than its omission, particularly when it is infused rapidly or in large quantities.[10] These complications include: (1) life-threatening hypokalemia; (2) tissue hypoxia, due to an increase in the affinity of hemoglobin for oxygen secondary to decreased red cell 2,3-diphosphoglycerate (2,3-DPG) content, which occurs following correction of the protective effect of acidosis; (3) increased plasma osmolality, and (4) central nervous system deterioration associated with elevation of plasma pH accompanied by an exaggerated fall in cerebrospinal fluid pH.

Because severe acidosis may result in diminished functions of the respiratory center and the myocardium, we administer 3.3 ml per kg of body weight of 7.5 percent sodium bicarbonate solution intravenously *over 12 hours* only in patients with severe diabetic ketoacidosis ($pH \leq 7.1$). The blood pH and carbon dioxide content are monitored frequently and administration of sodium bicarbonate is discontinued when the pH exceeds 7.1. We have not observed any harmful effects while following this approach. It is important to note that with the administration of insulin and fluids alone, the serum concentration of bicarbonate is being restored to normal.

Kaufman and his co-workers recommend the use of Ringer's lactate for correction of fluid and electrolyte deficits of their patients despite their strong arguments against the use of sodium bicarbonate. It should be emphasized, however, that approximately 28 mEq of bicarbonate result from each liter of Ringer's lactate.[12] Consequently,

in effect, sodium bicarbonate is being given. The administration of Ringer's lactate may have the same potential for complications as does sodium bicarbonate.

The administration of potassium in the form of potassium phosphate is also advised by Kaufman and his co-workers. This recommendation is logical because phosphate depletion is significant in diabetic ketoacidosis. The consequences of hypophosphatemia are partly reflected by deficiency of 2,3-DPG. However, the need for supplemental phosphate in the treatment of diabetic ketoacidosis has not been thoroughly investigated and phosphate solutions have usually not been administered to such patients at CHLA.

Acute cerebral edema has been an almost uniformly fatal complication of diabetic ketoacidosis in children and adolescents.[1,2,10] Although its pathogenesis is not fully understood, rapid return of the blood glucose concentration and pH to normal, increase in the polyol-pathway activity, insulin-induced alterations in intracellular binding of electrolytes in brain and excessive rates of intravenous fluid administration have been implicated.[1,2,10] It is probable that no single factor is responsible. It is our practice to administer glucose-free solutions if the blood glucose level exceeds 500 mg per 100 ml, to administer 5 percent glucose when the blood glucose concentration is less than 500 mg per 100 ml, and to give 10 percent glucose when the blood glucose level falls to 250 mg per 100 ml or less. No instance of fatal cerebral edema due to diabetic ketoacidosis has occurred at CHLA since 1965 following our treatment regimen.[10] Hypoglycemia resulting in prolongation and exacerbation of the ketoacidotic state has also been avoided by these recommendations. The rate of fall of plasma glucose was the same when the results of smaller doses of insulin were compared with results of larger doses.[7,11]

The goals of treatment of diabetic ketoacidosis are to control the diabetic state promptly with insulin and to restore deficits of water and electrolytes with the administration of intravenous fluids and electrolytes. In the hands of experienced physicians, the use of "small" or "large" doses of insulin appears to be equally effective in achieving a satisfactory outcome. Regardless of the insulin regimen, hypoglycemia and hypokalemia may occur. Blood glucose, potassium and pH must be measured frequently to assess whether the amount and rate of administration of glucose, fluid, electrolytes and insulin require readjustment. The successful management of diabetic ketoacidosis appears to depend more on the quality of care and how well therapy is individually tailored to each patient than on any specific form of treatment currently available.

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Force and Counterforce

THE HARSHNESS of Secretary Califano's recent statement before the AMA House of Delegates in San Francisco sharpens further the adversary relationship which has been developing for some time between medicine and the health care system on the one hand, and our government on the other. The Secretary implied nothing less than that the federal government has determined to use whatever force is necessary to bring the medical profession to the federal heel and health care costs to federal control. His assumptions appeared to be that the federal government has the power to do this, that there will be no opposition to doing it (except from doctors who number only one in a